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LETTERS TO THE EDITOR

Scope

Heart welcomes letters commenting on papers published in the journal in the previous six months. Topics not related to papers published earlier in the journal may be introduced as a letter: letters reporting original data may be sent for peer review.

Presentation

Letters should be:

- not more than 600 words and six references in length
- typed in double spacing (fax copies and paper copy only)
- signed by all authors.

They may contain short tables or a small figure. Please send a copy of your letter on disk. Full instructions to authors appear in the July 1996 issue of Heart (page 93).

Evaluation of mortality and morbidity during four years after late thrombolysis in acute myocardial infarction

SIR,—The effect of late coronary reperfusion after acute myocardial infarction (AMI) remains contentious.1 Coronary angiography is the gold standard for determining vessel patency; however, routine early catheterisation after thrombolytic therapy is not practical for most patients with AMI. Continuous ST segment monitoring¹⁻³ and imaging with 99mTc-labelled Sestamibi14 are useful markers of successful reperfusion and coronary artery patency after thrombolysis. In a preliminary study we found lower 3 year mortality in 87 AMI patients thrombolysed within 20 hours than in 65 AMI patients treated with heparin alone.5

In present study to determine whether late patency of the infarct-related artery affects the mortality and morbidity in AMI, we studied 228 patients (150 males and 78 females, aged 57 (5)) treated with alteplase (rt-PA) (group 1: n = 155) or heparin (group 2: n = 73). Patients were eligible if they were < 70 and their duration of pain was > 6 h (from 6.5 to 20 h after onset of symptoms). The efficacy of rt-PA thrombolysis, in terms of patency achieved, was assessed by non-invasive clinical criteria: continuous ST segment monitoring, early MB-CK peak (< 20 h), imaging with 99mTc-Sestamibi, sudden decrease in chest pain, and serial changes in abnormal wall motion. Four-year mortality was 6.5% in rt-PA patients (group 1) and 16.6% in those given heparin alone (group 2 v group 1, P < 0.01).

Reinfarction; need for bypass surgery or PTCA or admission to hospital; symptoms of angina pectoris and congestive heart failure; return to work; and the requirement for various medications during the four years did not differ significantly between the groups (P = NS). However, the cumulative

frequency of patients with ST depressions of > 1 mm at < 100 w of exercise and MIBIdefects was 12% in group 1 and 31% in group 2 (P < 0.05).

Our results show that late thrombolysis with rt-PA in AMI reduced four year mortality but did not have any effect on aspects of morbidity. This study supports the concept that thrombolytic therapy reduced the incidence of residual ischaemia postinfarction, although it was given late (6.5-20 h) after onset of symptoms.

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Catheter ablation for successful management of left posterior fascicular tachycardia: an approach guided by recording of fascicular potentials

SIR.—We read with great interest the paper Katritsis et al (Heart 1996;75:384-8) on catheter ablation of the left posterior fascicle for fascicular tachycardia (FT).

We have successfully used radiofrequency energy to the left posterior fascicle to treat two patients with FT. We confirm the observations made. We did, however, approach the left ventricular septum using a trans-septal technique and we believe that approaching the septum through the mitral valve allows better catheter manipulation and stability. One of our patients was treated during fascicular tachycardia. This should be considered as an alternative approach to the retrograde one described by Katritsis et al.

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Is thrombolytic therapy really better than conventional treatment in acute inferior myocardial infarction?

SIR,—Without wishing to prolong the debate on the relative benefits of thrombolytic therapy for patients presenting with inferior myocardial infarctions, we would like to make a correction of fact to Dr Tobé's reply

to our letter in the British Heart Journal (1995:74:476-478). The Fibrinolytic Therapy Trialists (FTT) overview clearly reports that 39% of patients (which we rounded up to 40%) of those forming the group who may be eligible to receive thrombolytic therapy are patients with ST elevation inferiorly, and not the 28% of patients to which Dr Tobé has corrected us.1 The FTT figures show a total of 58 600 trial patients. These are sub-grouped as: bundle branch block (2146), ST elevation anterior (13 229) ST elevation inferior (16 203), other ST elevation (10 187) (potentially thrombolytic eligible); ST depression (4237), other abnormality (9691), normal ECG (2907) (thrombolytic ineligible). Hence ST elevation in the inferior leads (16 203) is 39% of the potentially thrombolytic eligible group (total 41 765).

Readers can consider the correspondence on the interpretation of the data as presented by ourselves and Dr Tobé and come to their own conclusions on the appropriateness of thrombolytic therapy in patients presenting with inferior ST elevation who represent 40% of patients who are potentially eligible on the basis of the FTT overview.

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1 Fibrinolytic Therapy Trialists (FTT) Collab-orative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all ran-domised trials of more than 1000 patients. Lancet 1994;343:311-22.

Distress correlates with degree of chest pain: a description of patients awaiting revascularisation

SIR,-I congratulate Bengtson et al for establishing that fear and uncertainty are important symptoms in patients awaiting revascularisation procedures.1 We too identified fear and uncertainty in patients during a stressful period when they had more silent myocardial ischaemia on ambulatory ST Holter monitoring (together with a concomitant increase in urinary excretion of cortisol and noradrenaline) than they did during a later (non-stressful) period.2 Fear and uncertainty are therefore relevant as independent risk factors in ischaemic heart disease.

Anxiety and depression, both significantly associated with pain in Bengtson's study, have already been identified as important precursors of further ischaemic events and sudden cardiac death.3 Though it is true that dyspnoea may indeed reflect heart failure, as discussed in Bengtson's paper, hyperventilation is a frequent concomitant of fear and anxiety and has been documented in patients with angiographically proven coronary heart disease.4 Because it not only confounds the clinical picture (viz dyspnoea and increased reports of chest pain) but also lowers the threshold to ischaemia,5 hyperventilation should be looked for and adequately treated.

There is now substantial evidence that

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psychosocial factors, which include social isolation, degree of control and job strain, depression and loss of well-being, together with psychological characteristics of phobic anxiety and anger/hostility⁶ should be assessed as assiduously as smoking, cholesterol, and hypertension, and appropriate treatment initiated.

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NOTICES

The Second International Meeting of the Working Group on Heart Failure (Heart Failure '97) will be held on 24-27 May 1997 in Cologne, Germany. The meeting is organised under the auspices of the European Society of Cardiology. For further information please contact Dr Willem J Remme, Sticares Cardiovascular Research Foundation, PO Box 52006, 3007 LA Rotterdam, The Netherlands (tel +31 10 485 51 77; fax +31 10 485 48 33.

The 12th Symposium on Echocardiology and the 9th Meeting of the International Cardiac Doppler Society will be held on June 25, 26, and 27 1997 at the Erasmus University Congress Centre in Rotterdam, The Netherlands. General information can be obtained from LMC Congress and Business Services, PO Box 593, 3700 AN Zeist, The Netherlands (tel: +31 (343) 515 134; fax: +31 (343) 533 357; e-mail: http://www.eur.nl/FGG/THORAX/ECHOD OPPLER).